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Excitotoxicity, free radicals, and cell membrane changes.

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Neuronal injury resulting from glutamate receptor-mediated excitotoxicity has been implicated in a wide spectrum of neurological disease states, including ischemia, central nervous system trauma, and some types of neurodegenerative diseases. Excitotoxicity may interact with other pathophysiological processes to enhance neuronal injury; for example, excess glutamate release due to free radicals generated during the immune response to infection might initiate secondary excitotoxicity, and intracellular pathways that contribute to neuronal destruction may be common to both excitotoxic and nonexcitotoxic injury processes. Defining the contribution of excitotoxicity to neuronal damage in acute zoster infection and post-herpetic neuralgia may provide one means of reducing morbidity from this often debilitating disease.

Publication Types:

- Review
- Review, Tutorial

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